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THE SEPARATE LOCALIZATION IN THE CORTEX AND SUBCORTEX OF THE CEREBRUM OF THE REPRESENTATION OF MOVEMENTS AND OF MUSCULAR AND CUTANEOUS SENSIBILITY.*

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In 1888, and on several occasions since, the subject which is the basis of my remarks today was discussed at meetings of this Association. The weight of opinion in the Association, as expressed in these discussions, seems to have been against the position which I have uniformly taken, and with but slight modification have consistently held. At the outset it may be well for me to restate this position: I hold to the separate localisation in the cortex and subcortex of the cerebrum of the representation of movements and of muscular and cutaneous sensibility, not believing that the Rolandic and adjacent regions are sensori-motor as has been held by Munk, Hitzig, Bastian, Dana, Starr, Dejerine, and others;

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that the cerebral motor zone includes only the precentral, postcentral, and paracentral convolutions, and the caudal extremities of the first, second and third frontal convolutions, and the anterior fourth of the superior parietal convolution as usually represented; and that a separate and extensive sensory zone exists, which includes the gyrus fornicatus, precuneus, and postparietal convolutions, possibly also the hippocampal convolution as held by Ferrier. With regard to the subparietal or inferior parietal convolution, the evidence at hand is not as conclusive as for the other gyres included by me in the sensory zone. My views are indicated semi-diagrammaticaly in Figs. 228 and 229, page 333, of my "Treatise on Diseases of the Brain and Cranial Nerves." The main object of the present remarks is to show that additional facts supporting this position are now at our command, and a special reason for again bringing the subject before the Association is to be found in the fact that several of the contributions to the proceedings of this meeting contain evidence in favor of the doctrine of separate sensory and motor localization. My paper is not intended to be a recital of original observations or a study of the literature of the subject, and yet I trust it will be seen that it is based on both. The recent work to which I shall appeal as upholding the view of the separate representation of movement and of cutaneous and muscular sensibility is entirely that of members of this Association, and may be included under the following heads: (1) Observations on sensory phenomena and especially on astereognosis in connection with cases in which operations guided by localizations have been performed; (2) clinical observations with autopsies and microscopical investigation showing the existence of the *carrefour sensitif* of Charcot; (3) the case of Spiller in which was demonstrated the extent of the motor cortex in man from the cortical degeneration present in amyotrophic lateral sclerosis, the region thus mapped out corresponding closely to the motor zone as given by von Monakow and by the writer; (4) new observations on cases in which operation and autopsy have demonstrated that lesions strictly limited to the motor zone, or to this zone and the region anterior to it, have shown that no objective sensory phenomena were present; and (5) the record of a case of softening in which astereognosis and ataxia were present, but not motor paralysis.

The cases of brain tumor recently recorded by me are confirmatory of the view that the different forms of muscular and cutaneous sensibility have a cerebral localization separate from that of the motor area.[1] They are of special interest in connection with the study of astereognosis. In all the cases in which disorders of sensibility including astereognosis were present, the evidence pointed to the fact that the lesions accounting for these symptoms were located in the postparietal region—the region between the retrocentral and parieto-occipital fissures. The conclusions arrived at were that tumors of the postparietal region, and especially of the superior parietal lobule (parietal of Wilder), give as their most important localizing symptoms disorders of cutaneous and muscular sensibility, and especially astereognosis; other symptoms often present in such cases are the result of compression or invasion of adjoining regions. In referring again to the reports of these cases and the conclusions drawn from their study, I wish to emphasize the fact that at certain stages in some of them true motor paresis was absent.

In addition to the cases observed by me other cases have been recorded, as by Walton and Paul,[2] and by Burr,[3] in which astereognosis and some of the so-called forms of cerebral anesthesia have been present without motor paralysis, in contradiction to the assertion of Verger[4] that he knows of no case in which cerebral anesthesia existed without motor paralysis. The long-since recorded case of Starr and McCosh[5] seems to be overlooked by the more recent writers on this subject. It is true that the stereognostic sense in the recent method of regarding this subject was not studied, but the record shows distinct loss of muscular sense without motor paralysis. Let me cite here a brief summary of this case, which was, the reporters say, "comparable to an accurate physiological experiment upon the cortex of the brain, although no such experiment was intended. It was one of traumatic epilepsy characterized by psychical attacks and headache. Trephining and the removal of a small angioma were followed by loss of muscular sense in the right arm lasting six weeks. The operation was performed upon a spot of the brain about the junction of the superior and inferior parietal convolutions, clearly posterior to the postcentral convolution, and resulted in a loss of muscular sense in the opposite hand and forearm, without any disturbance of other sensations or of movement."[6]

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[1] Mills, Keen, and Spiller, JOUR. NERV. AND MENT. DIS., v. 27, May, 1900. Mills, Phila. Med. Jour., April 20, 1901.

[2] Walton and Paul, JOUR. NERV. AND MENT. DIS., v. 28, April, 1901.

[3] Burr, American Jour. Med. Sci., v. 121, March, 1901.

[4] Arch. Gén. de Méd., Nov. and Dec., 1900.

[5] American Jour. Med. Sci., v. 108, 1894.

Walton and Paul have advanced an ingenious explanation of the cerebral sensori-motor mechanism, comparing it with the mechanism of speech. While agreeing in large part with these writers, I do not fully concur with their views, and the difference is essential. They suggest (1) that we may have the cortical structures receiving impressions of simple touch, temperature and pain, locating these centers as they have been located by the writer and others holding similar views; (2) that we may have a second mechanism for the associative memories resulting in the appreciation of the character of the object felt; and (3) centers assembling the stimuli from the various groups of the second order and also from other sources. If I understand them correctly, this third or assembling center is placed in the Rolandic or motor region proper, and it is with this portion of their suggestion that I do not agree. With them, and differing from Dana, I believe that it is logical and in accordance with analogies to believe that the cerebral sensory mechanism is compound and even complicated. Stated as simply as possible, my view of the cerebral sensori-motor mechanism is that we have (1) certain perceptive centers for cutaneous, muscular and other sensations comparable to the well-known visual and auditory perceptive centers; (2) that we have associative centers perhaps comparable with the speech concept centers, and (3) that we have a higher stereognostic center where takes place the final assemblage and arrangement of the stimuli which are to call forth movement by evoking the cells of the entirely distinct motor or executive region. This stereognostic center is to be compared with the propositionizing center of Broadbent in Broca's convolution, and not with the centers which contain the executive cells concerned with motor speech at the feet of the central convolutions. Even Walton and Paul, while they hold that it is a reasonable supposition "that the Rolandic region contains at least the areas representing the principal aggregation of cutaneous and muscular memories whose correlated action acquaints us with the various qualities of external objects," admit that the Rolandic region is not the sole seat of sensation. Burr places the stereognostic center in the superior parietal convolution, where I had previously located it, although it is not yet demonstrated that it is limited to this convolution. The time has not yet arrived for the exact anatomical subdivision of the sensory zone or region, but some facts, anatomical, physiological and clinico-pathological, seem to indicate that cutaneous sensibility has its centers of representation chiefly in the limbic lobe, while muscular sensibility and the stereognostic sense are more especially represented, as has already been stated, in the postparietal region, which includes the superior and inferior parietal convolutions and precuneus. Should this prove to be the case, the losses and disturbances of cutaneous sensibility found in lesions of the two great parietal convolutions are probably to be explained by a cutting of sensory tracts on the way to the limbic lobe.

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[6] Mills, "The Nervous System and Its Diseases," Phila., 1898.

A word might be said here with regard to Bastian's[7] view of kinesthesis and kinesthetic centers. As defined by Bastian, kinesthesis, or the sense of movement, is included in part at least in the stereognostic sense. It has been used to include all those impressions which are evoked by muscular movements, including "cutaneous impressions, impressions from muscles, fasciæ, tendons and articular surfaces, and in addition a set of unfelt or but little felt impressions which guide the activity of the brain by the information (unconscious) which they afford us as to the different degrees of contraction of all the muscles concerned in the production of movement." (Bastian.) While recognizing the existence of such a sense, I believe that its representation should be placed not in the motor, but in the postmotor region.

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[7] Bastian, "The Brain as an Organ of Mind," N. Y., 1883.

An acceptance of Charcot's *carrefour sensitif* favors the doctrine of the existence of separate areas of representation for cutaneous and muscular sensibility, for if a tract which conveys these forms of sensibility is compacted together in a special and limited portion of the internal capsule, it is probable that the upward extensions of this tract pass to special cortical regions. The opponents of the theory of separate sensory localization would seem to accept this way of looking at the matter. Dejerine, for instance, one of the strongest advocates of a mixed sensori-motor cortical zone, holds also that motor and sensory fibers are commingled in the internal capsule. A marked tendency has been shown in recent years to abandon Charcot's idea of the sensory cross-roads, in spite of the old facts apparently demonstrating its existence. The belief in the existence of such a tract is not in necessary antagonism with the view that the thalamus is a great basal center for sensation. It is probable that large portions of the sensory tract pass to the thalamus, and that other sensory fibers go directly to the cortex. It is also probable that fibers conveying sensation pass from the thalamus to the cortex. Just where these different bundles end is yet to be determined, but I do not believe that they pass, or even that they mainly pass directly to the motor zone. The contribution to this meeting by Drs. Dercum and Spiller would seem to be a final confirmation of the existence of the *carrefour sensitif*. I had the opportunity of seeing and studying the patient referred to by them in the wards of the Philadelphia Hospital as early as 1892.

The case was included in a series of cases of hemianopsia recorded by Dr. de Schweinitz and the writer in Vol. III of the "Philadelphia Hospital Reports" (1896), where it was recorded as a case of right lateral quadrant anopsia, with absence of Wernicke's symptom; but with dyslexia, right hemiparesis, partial right hemianesthesia and partial word-deafness and word-blindness. The case as recorded by Dr. Dercum[8] was one of hemiplegia with exaggerated reflexes associated with hemianesthesia, also with right homonymous hemianopsia. As reported by Dercum and Spiller, autopsy and careful examination were eventually made on this patient. The autopsy showed that the primary lesion was confined to the extreme posterior portion of the posterior limb of the internal capsule. The thalamus was not invaded by it, although tracts of secondary degeneration were traced into this body.

The results of embryological studies in myelinization by Flechsig and others are not in antagonism with the view that there are separate areas of representation for sensation, movements and for the assembling processes which intervene between sensation and movements, although some of these may at first sight appear to be opposed to this view.[9] I shall omit any reference to centripetal neurone systems below the level of the internal capsule and red nucleus. Flechsig traces the upward continuations of neurone systems both of the lemniscus and the red nucleus, although he does not discriminate between these two sets of fibers in his descriptions. He divides all the central centripetal neurones after they have reached the level of the red nucleus and internal capsule into three systems of fibers which become medullated at different ages and can therefore be distinctly separated from each other.

The first of these systems is medullated at about the ninth fetal month, occupies the most posterior part of the internal capsule, and in its upper half the area immediately behind the pyramidal tract. Its fibers coming in part from the median lemniscus are distributed according to Flechsig, exclusively to the cortex of the two central convolutions.

The fibers of the second system which come entirely from the thalamus and chiefly from its lateral nucleus, become myelinated one month later than those of the first system, and while one portion of this system passes to the convolutions included by me in the motor zone, the main body of the fibers is distributed in three separate bundles to the gyrus fornicatus, hippocampal convolution and uncinate convolution, in brief, to the entire limbic lobe.

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[8] Dercum, Jour. Nerv. and Ment. Dis., v. 26, April, 1899.

[9] These views are summarized from L. F. Barker's "Nervous System and its Constituent Neurones." N. Y., 1899. In this work numerous references to the investigations of Flechsig will be found.

A third system which is medullated still later emerges from the anterior portion of the lateral nucleus of the thalamus, passes into the internal capsule about its middle portion, then subdividing is eventually distributed by more or less separate bundles to the third frontal convolution, the middle portion of the gyrus fornicatus, the anterior portion of the superior or first frontal convolution and the foot of the middle or second frontal convolution.

It is altogether probable that the fibers of those systems which are distributed to the limbic lobe are concerned with the transmission of sensations which have their cortical termini in this lobe. Those fibers which go directly to the convolutions of the motor region are concerned with the more direct cerebral reflexes.

The views here presented with regard to the sensori-motor mechanism accord in the main with the ideas involved in Flechsig's theory of association centers. Both the associative centers comparable to the speech concept center and the higher stereognostic or assembling center would probably be included in Flechsig's association centers. The superior and inferior parietal convolutions and the precuneus form an important portion of his associative system, and in these convolutions the clinical facts here presented would seem to indicate that the associative and stereognostic centers are situated. A little consideration will make it evident that the facts of clinical medicine are in accord with those of embryological research. The muscular sense and especially the stereognostic sense as it is now understood, are higher developments than the senses of touch, pain and temperature. Flechsig has shown that the white matter in the superior and inferior parietal lobules, like that of his other association regions, becomes medullated considerably later than that of the sense centers, those in the limbic lobe, for instance. These association centers furnish a mechanism which makes possible "the working up into higher units of simple sense impressions and of combinations of simple sense impressions of the same qualities and of different qualities." (Barker.)

In this connection I wish by reiteration to make my position entirely clear. I believe that the centers both for the more simple forms of sensibility like those of touch, pain and temperature; and the centers for such higher processes as stereognosis, are entirely distinct from the motor centers proper.

The case of Spiller[10] in which he determined the extent of the motor cortex in man from the degeneration of amyotrophic lateral sclerosis is in my judgment so far as it goes confirmatory of the views held by me regarding separate sensory and motor localization in the cerebral cortex. The diagram obtained by Spiller conforms quite closely to that given by von Monakow and by the writer for the cortical motor area. Spiller examined blocks of cerebral tissue removed from the Rolandic and adjoining regions, and stained these by the Marchi method, and from the results obtained in this way his diagram was made. He says that the portion corresponding to Broca's region shows the presence of degenerated fibers. He was not able to observe degeneration of the gyrus fornicatus below the paracentral lobule, or on the median aspect of the first frontal convolution. No degeneration was found in the parietal lobe from a point a little posterior to the upper extremity of the postcentral convolution. A study of his diagram shows that the gyrus fornicatus, precuneus, superior parietal lobule in the main, and the inferior parietal lobule are not included in the motor region. The areas excluded are in other words those which have always been regarded by me as not motor in function.

The fourth head under which was included the recent work upholding the view of separate sensory localization was that of newer observation on cases in which operation or autopsy, or both, have demonstrated that lesions strictly limited to the motor zone, or to this zone and the region anterior to it, have shown that no objective sensory phenomena were present. A number of cases of this sort might be cited, but I shall refer to only two, both reported at this meeting. One of these cases, briefly referred to by me in a previous paper,[11] is recorded at length by Dr. Sinkler.

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[10] Spiller, "Contributions from the William Pepper Laboratory of Clinical Medicine," Phila., 1900.

My views as to general diagnosis and location were confirmatory of those held by Drs. Sinkler and Potter, namely, that the lesion was a tumor, and was largely, if not entirely, confined to the motor zone. The patient had no objective sensory symptoms, although she at times complained of a numb feeling in the left side of the face, left arm and left leg, which were paretic, the loss of power being marked in the lower extremity. She had several convulsive seizures chiefly affecting the limbs of the partially paralyzed half of the body. All the deep reflexes on the left side were increased, persistent ankle-clonus being present. The Babinski phenomenon was elicited on the left side. Headache was not conspicuous, but vomiting occurred, and double optic neuritis was present. The most notable point is that objective symptoms referable to the areas of muscular and cutaneous sensibility and the special senses were absent, excepting of course the optic neuritis, which has no localizing value. In this case the autopsy showed the tumor compressing the motor region.

The second case to which I desire to make reference is that of Leszynsky and Glass.[12] It is sufficient for the purposes of my argument to say that in this case the tumor was located in the motor region by motor symptoms; that the growth was successfully removed, and that even the post-operative symptoms were purely motor. A study of the different forms of sensibility was made, but without revealing any sensory symptoms.

The last evidence to which I shall allude in favor of the position taken in this paper is that afforded by a case very recently observed. A middle-aged woman was seen twice by me at the Woman's Hospital in Philadelphia. She had no history of syphilis, but one of malaria, malarial parasites having been found in her blood some weeks before my examination of the patient. The case was also seen in consultation by Dr. Spiller, who was present at the autopsy. I shall omit all points except those bearing on the question of localization. The chief symptoms and signs were optic neuritis of high grade, pain in the occipital region, mental hebetude increasing, stiffness of the neck with retraction of the head, ataxia and astereognosis of the left upper extremity, nystagmus on lateral movement, and sluggish knee-jerks. No true motor paralysis was present. The ophthalmologist had not recorded any examination of the visual fields, and the condition of the patient precluded our determining the presence or absence of hemianopsia. Autopsy showed a large area of softening which to the naked eye was confined to the superior parietal lobe, not extending into the central convolutions. The autopsy was held only a few days before this meeting, so that a sufficient time has not elapsed to allow of a more careful report on the case.

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[11] Mills, Phila. Med. Jour., Apr. 20, 1901.

[12] Leszynsky and Glass, Medical Record, Sept. 28, 1901.

I do not intend in the present paper to go over at length the older arguments for and against separate sensory and separate motor localization in the cerebrum. These can be found in my work on diseases of the brain and cranial nerves, and in Schäfer's new "Text-Book of Physiology" (Edingb., Lond. and N. Y., 1900, v. 2), but I may be permitted a brief glance at the most important of them.

Physiological experiments have shown that destructive lesions of various parts of the limbic lobe cause anesthesia, especially loss of the pain and temperature senses, and in at least a few cases lesions of the gyrus fornicatus have resulted in permanent anesthesia. The well-known fact that lesions of the lateral aspect of the cerebral hemisphere, and especially of the motor region far outnumber those of the limbic lobe and other portions of the mesal aspect of the cerebrum, explains in part the comparatively few clinico-pathological cases demonstrating anesthesia from lesion of the purely sensory zone.

In the encircling experiments in which the motor region was isolated from the rest of the brain, the motor cells would not respond to tactile or other sensory impressions although they would to direct excitation.

When removal of blocks of the motor cortex was carefully done the experiments were not followed by anesthesia.

It is only necessary to refer to the numerous recorded cases of lesion of the motor cortex without impairment of sensibility, and also to the cases in which excision of the human cortex during surgical operations has likewise not resulted in anesthesia.

The strongest case made out in favor of a Rolandic sensori-motor cortex is that which has been built up from a study of cases of lesion in this region, or alleged to be in this region, in which sensory as well as motor phenomena have been present, but if every such case were carefully analyzed a satisfactory explanation of the apparent contradiction would be forthcoming. I shall recall here a few of these explanations. Some of these sensory disorders are hysterical or at least functional. Subjective sensory disturbances should not be given weight, as these may be present in pure motor paralysis, and are largely the result of suggestion. Circulatory disturbances in parts beyond those included in the paralyzing lesion account for some such cases, according to Legroux and de Brun,[13] and pressure on neighboring parts accounts for the impairment of sensation in other cases. Defects of sensation are sometimes due to local causes in the paralyzed limbs, as to extreme coldness or lymph accumulation. Another argument made use of by Schäfer is not given the weight which it deserves. In a few words it may be expressed by saying that as all cerebral sensory areas are connected by association fibers with the motor area, it follows that a lesion in the motor area will sever this connection. The effect of such severance may be felt for a time at least, not only in the motor area and its projections, but also in the parts from which it is isolated, namely, in the sensory and association regions; and not only is this true, but a real degeneration or involution of those parts of the nerve fibers which are proximal to the lesion occurs, hence we may have both temporary and even persistent sensory phenomena as the result of a lesion not in the sensory sphere.

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[13] Legroux and de Brun, "L'Encephalon," 1884.

Owing to the fact that the motor and sensory regions are co-terminous, lesions mainly in one region frequently invade to some extent the other. Every case should be excluded from a collection intended to prove that the Rolandic region is sensori-motor, in which the lesion described extends even for the fragment of an inch into any part of the great encircling sensory region. The greatest care should be taken by those who collect data of this kind to accurately describe the exact limits of the lesions. It will, I believe, be found that very many cases included in the well-known collections of cases used by the advocates of a sensori-motor zone, are not cases in which the lesions are strictly limited to the motor zone as described in this paper. All cases of recent operation on the motor zone or of recent hemorrhage into this region should be carefully sifted before they are accepted as evidence. By pressure, bruising and infiltration, the influence of lesions of this character is usually extended beyond the area of the lesion, or what is supposed to be this area.

DISCUSSION.

Dr. G. L. Walton said that there is one thing we must remember in connection with an investigation of separate cases. A great many individual cases have been produced as absolutely proving this or that, but a very little experience with the study of these cases will convince us that we cannot draw definite conclusions from any single case, or any half dozen cases. By definite conclusions he meant deciding, because we find a lesion in a certain region, that this region is the center of the function which is lost. Dr. Mills himself had elaborated on this subject particularly with regard to operative cases. To realise this fact we only need to remember the effect of operation upon distant parts of the brain with whose function we are familiar, for example, Dr. Walton had seen a case in which a mere bit of cortex was removed from the upper Rolandic region, no further damage being done, as far as one could see, but after operation the patient had mind-blindness and word-deafness. We certainly would not draw the conclusion that these functions have their seat at the upper end of the fissure of Rolando. When we come to study cases in which no symptoms were produced before operation by a tumor in the Rolandic region or elsewhere, and to study the symptoms that took place after operation, which set of symptoms are we going to attribute to that locality? Dana quotes a case in which a large tumor was found in the Rolandic region which had produced no symptoms before operation, but after operation most extensive loss appeared; including sensations of pain, touch and temperature as well as the stereognostic sense. Now, personally, in a case like this, if either set is regarded as final we should choose the ante-operative symptoms, for these, if any, symptoms are to be attributed to the lesion itself. Dr. Walton was arguing against himself a little, because this case would indicate that the Rolandic region is free from sensory function, a view with which he did not agree. We all know, however, that a tumor may be found in a certain situation which we recognize as the seat of a certain function, and yet the function not be lost. This only illustrates the fallacy of depending on single cases. What we can do is to study groups of cases. He had recently looked over the carefully reported cases, among Dana's last 25 cases, and the cases of Verger, and he had found that the stereognostic sense is often lost alone in lesion of the central convolutions, whereas cases in which both the fissure of Rolando and the parietal region are affected are apt to lose not only the stereognostic sense, but also pain, touch and temperature. If we may draw any conclusion from such a study, we should draw the conclusion that the stereognostic sense is situated in the Rolandic region, and pain, touch and temperature more posteriorly. But he did not claim for a moment that the study of one group of cases proves even this fact. He merely said that such study is suggestive, and that if we start out with the preconceived idea that this is the distribution, we shall be surprised to find how the cases fall in line, as it is equally true if a person starts out with any preconceived idea he can fit almost any case to his theory. The same cases have certainly been adduced more than once to fit different theories. There seems to be very little difference between Dr. Mills' present position and the position recently advanced in the paper by Dr. Paul and Dr. Walton to which Dr. Mills had alluded. The only difference as Dr. Walton understood it is that instead of placing the stereognostic sense in the Rolandic region Dr. Mills places it a little more posteriorly. Dr. Walton agreed with Dr. Mills that Broca's convolution is not a motor center in any sense of the word, and that the stereognostic center, if there be such an one, is not a motor center. From his point of view the stereognostic center represents an aggregate of memories of touch, pain, temperature, sights, sounds, and even memories of the words which are used in connection with the movements we are about to make, and the objects we handle. Now, no one can absolutely say, and no one will, perhaps, ever absolutely be able to say, whether this aggregation takes place in the Rolandic gyri or the anterior part of the parietal lobes. Perhaps Cajal has led the way to solving the problem in his recent study of the anatomy of the cortex, in which he shows very clearly that there is a distinct difference in the formation of the posterior and the anterior Rolandic gyri; whereas the anterior Rolandic gyrus has many large and middle-sized pyramidal (motor) cells, there are many less of these in the posterior central gryus and an entirely different set of nerve fibers, nerve plexuses, giving the associative cortex type. Cajal suggests that there is either a different kind of motion carried on posteriorly, or perhaps, that the posterior gyrus is sensory and the anterior motor. The latter suggestion would fit very nicely with the supposition that the stereognostic sense is situated in the posterior Rolandic gyrus and motion in the anterior; Dr. Walton agreed with Drs. Mills and Dercum that the sensory and motor functions are separate. Certain cases have been reported. Dr. Walton had seen two or three cases in which sensation was lost without motion; these cases are so few that Dana practically denies them and Verger regards them as very exceptional. We may certainly draw the conclusion that these centers, though distinct, are very close together. Dr. Walton remembered one case of Rolandic lesion in which the stereognostic sense was at first lost without loss of motion, but in a few days loss of motion followed. The centers may overlap. Perhaps, as Horsley suggests, the small cells are sensory and the large cells motor, in which event the sensory cells might easily overlap the motor cells which are to carry out the action which has its concept in the sensory cells. Dr. Walton thought that Dr. Mills has to explain away many cases of Rolandic lesion, with loss of stereognostic sense, by assuming that a different region is implicated, and it seems more reasonable to attribute this function to the central convolutions.

Dr. P. C. Knapp said it was somewhat interesting to hear Dr. Mills change his position from that which he held six years ago when he admitted that the sensory center extended as far forward as the fissure of Rolando. He had apparently gone back half an inch if not more. At that time Dr. Mills admitted that the center did extend to the fissure of Rolando. The clinical evidence, however, in favor of sensory distribution up to, if not anterior to the Rolandic region seemed to Dr. Knapp very strong, as he had held when he read his paper upon the subject eleven years ago before this Association. The evidence, however, as to a center for tactile gnosis or stereognosis is as yet somewhat indefinite. *A priori* we must admit that stereognosis is a cortical function, yet we can not determine the exact center for that cortical function on the evidence of many of the reported cases any more than we can determine the evidence in favor of a center for visual gnosis or auditory recognition in those with defective hearing or sight. We must find that the patient has absolutely normal sensibility in every other respect. We cannot diagnosticate visual agnosis until we demonstrate that the patient has a clear visual power. We cannot demonstrate that a patient has psychical deafness until we have demonstrated first that the patient has a perfect auditory faculty. So, until we have demonstrated that the patient has a perfectly normal tactile sensibility in all its forms, which is a most difficult process, we cannot demonstrate conclusively a center for stereognosis. We cannot be absolutely certain of our center for cortical recognition of tactile impressions, inasmuch as we know that in lesions of the sensory tract lower down we very often have from the defective sensibility defective recognition. Dr. Mills had, Dr. Knapp thought, overlooked to a considerable extent the developmental and anatomical relations of the sensory tract, the demonstration that the sensory fibers do pass into the Rolandic convolutions as well as into the parietal, and he had laid altogether too little stress upon the large number of clinical cases of lesion of the Rolandic region in which there had been very definite loss of sensory function.

Dr. F. W. Langdon said he had recently had under his care a case that perhaps should contribute a mite to this discussion. The patient, a married woman, 24 years old, with a child about a year old, for seven years had had severe epileptic convulsions, typical grand mal attacks with unconsciousness, and cry, and had bitten her tongue at various times. She had commonly two or three of these attacks a day, sometimes six, and rarely went a week without two or three except when under the influence of bromides given in enormous quantities. When she was brought to Dr. Langdon's office she was in a state of mild dementia from the administration of bromides for nearly a year. They did not control the attacks, but made them milder. She had numerous mild attacks. She was practically disabled. Dr. Langdon recommended that she be put to bed for a week or two and given diluents and liquid diet, with strychnia and arsenic. She came back inside of ten days a different person, bright and active, but the convulsions recurred. The attacks began in the left little finger with a twitching, and sometimes extended to the wrist and sometimes not. The finger or hand would twitch for half a minute, after which a generalized convulsion would often follow; and she would have three or four of these attacks daily. Sometimes twelve or fifteen of the mild Jacksonian attacks would occur in a day, even when the severe attacks were modified by the bromides. The bromides did not stop the Jacksonian attacks, but made the severe attacks milder. She had no defect of tactile or temperature or stereognostic sense in any way at the second examination, and the motor power was not notably deficient in the left hand. After watching her two or three weeks, Dr. Langdon advised the opening of the Rolandic area over the hand center. This was done by his colleague, Dr. E. W. Walker. The scalp was shaved and no evidence of a scar was found. At the operation, which included the removal of a two-inch trephine button, a thickened, softened, degenerated condition of the dura was found over less than one inch of that area and the dura was fused with the arachnoid and the pia and cortex, causing a distinctly limited localized pachymeningitis of degenerative character, the tissue so friable that it would scarcely hold a ligature. Nevertheless the membranes were separated as well as possible, the probe was passed under the dura, and everything was done to relieve the supposed irritation at that point. Perhaps more was done by the removal of the button of bone than anything else. The stereognosis before the operation on numerous occasions, was perfect, as she recognized coins, etc. Less than six hours after operation she had a severe convulsion which was the only one she had for the next three weeks; and then a second one was due to fright evidently. On the second day after the operation she had a marked weakness in the hand, and this advanced so that she could not grasp anything, and then the stereognostic sense having been tested she could not recognise anything, even things she was familiar with before, such as coins, pen-knives, etc. Astereognosis was absolute about ten days or two weeks, and then sensation began to come back along with motor power. Motor power came back first, and then the ability to recognize objects, and when she left the hospital four weeks after the operation, she had good use of the hand and good appreciation of tact, pain, temperature and stereognosis. Of course we cannot say because the operation was limited to a small area that the effusion was also limited to that area, but the case bears upon the question at present under discussion, and so far as it goes would locate the function of stereognosis as applied to that segment of the limb, in the same area as the motor power and ordinary sensation.

Dr. Wharton Sinkler said that in his case to which Dr. Mills referred there was a tumor as large as a hen's egg springing from the dura and dipping down into the Rolandic fissure, separating it and involving by pressure only the anterior and posterior Rolandic convolutions. In this patient there was no astereognosis, no disturbance of pain or temperature sense, and only mild motor phenomena. In Dr. Sinkler's experience astereognosis is more constantly associated with disease of the thalamus than with any other part.

Dr. Theodore Diller spoke concerning a patient under his care. In brief, this patient presented the general symptoms of brain tumor, that it to say, he had headache, optic neuritis, vomiting and had had a number of general convulsions. There was no motor paralysis, and no disturbance of common sensation, and no disturbance of heat, cold or temperature senses; but there was absolute astereognosis in his right hand. The right foot was tested—of course, tests could not be made so accurately in the foot as in the hand—and seemed to show no astereognosis. The question of localization might be decided by operation or autopsy or both very soon, and Dr. Diller thought he might have something further to report next year. In the mean time, he would be very glad indeed to have Dr. Mills tell him where to make the trephine hole.

Dr. James Hendrie Lloyd thought that every neurologist ought to try to get himself on record when it comes to a discussion of localization of sensation, especially the stereognostic sense. He wished to say merely this: A similar discussion had been held in Philadelphia some time ago. At that time Dr. Lloyd called attention to the fact that the stereognostic sense is a very different thing from the mere sensorimotor reflex function of the cerebral cortex; in other words, the sense of location in space, location of form, location of what we might call the distensible qualities of matter involves a distinct psychological function, and we have every reason to suppose from the standpoint of physiological psychology that such a physiological function should have some location in the cerebral cortex. He did not think we have any positive clinical or experimental grounds for saying that the mere tactile sense, as we know it, can be accurately localized in the cerebral cortex; but when we come to this very complex psychological function of distinguishing the qualities of space, there we have to suppose that there is an intellectual act which can only, according to our ideas of cerebral function, be localized somewhere in the cerebral cortex. As to this localization we have some clinical grounds for believing that it is localized in a certain area of the cortex. The case to which Dr. Mills had referred of brain tumor is now quite a classical one, one in which Dr. Lloyd was privileged to express an opinion before that case was brought to operation, and basing his opinion upon experience with a preceding case in his clinic at the Philadelphia Hospital, he expressed an opinion independently of all others that the lesion was probably in the superior parietal lobule, and there is where the operation accurately revealed that lesion. The precedent case upon which he based that idea was one which was not a tumor, but a cerebral hemorrhage breaking through and breaking up the cortex in the superior parietal lobule. In both those cases there was a very evident loss of the stereognostic sense. It seemed to him that it is merely logical to suppose that this intellectual function, this gathering together of sensory impressions and making out of them an intellectual concept which is necessary to localise objects in space and to localize their shape, and size and weight, must have a localization in the cortex, and that these clinical cases tend to show that that localization is in the superior parietal lobule. That is the position, so far as he could see it, which so far our clinical knowledge allows us to take.

Dr. Joseph Collins had very little to say about this matter at this time because he had had no experience to cause him to change his views of the subject which he had heretofore expressed before this Society. So far as his own experience went the sensory perceptual area of the brain coincides entirely and wholly with the motor area. He was not denying, however, that that portion of the cortex immediately behind and bordering the Rolandic area, the anterior portion of the parietal convolutions is not concerned as well with it. He believed upon the evidence that had been furnished by Dr. Mills and by others, that it is, but so far he had had no personal experience which led him to maintain that it is. Dr. Collins said that he could relate one or two cases which he had had the past winter, that have some bearing on this matter. One case he had operated on a few weeks ago in which a subcortical tumor, immediately subcortical, had not wholly pierced the cortex, and was in the Rolandic region and produced the most typical astereognosis that he had ever seen. He did not know whether there was any other lesion in this woman's brain or not. Apparently the astereognosis, as well as the Jacksonian epilepsy in this case, was dependent on the growth in the middle of the Rolandic region. He was firmly of the belief that there is in reality no center for stereognosis. He believed that he was in accord in this matter with Dr. Burr who says that stereognosis is not perceptional, but the sense is a conceptional one. This position he understood to be at variance with Dr. Lloyd's. Dr. Collins did not believe there is a single area in which this intellectual process is situated. He believed there are many areas of the brain through the activities of which we get perceptions, and apperceptions that contribute to stereognosis. One of these is the Rolandic area, another may be the parietal lobe, and another the frontal lobe. As he stated in the beginning, all he wished to say was to reiterate former expressed opinions which were in accord with the Chairman's and Dr. Dana's. At the same time he was impressed with the evidence that Dr. Mills had brought forward in support of his contention.

Dr. Hugh T. Patrick said that the matter must turn on the evidence of more facts; facts such as have been demonstrated today; that is, cases examined with scrupulous care, both before and after death, with particular reference to this question. So many of the previous cases have been observed not with particular reference to sensory disorder, and particularly not with special reference to the so-called stereognostic center. He was not prepared to take his stand with the extreme localizationists, but he though we must concede that there are some places in the brain which subserve sensation especially, and it might be in quite a restricted manner. Queerly enough, the day he left home he saw almost the counterpart of the case reported by Drs. Dercum and Spiller, of course without necropsy. The woman had practically no paralysis. There was very distinct sensory disturbance and hemianopsia. Last winter, he had under his care and under constant observation for a number of weeks, a case of what must be typical astereognosis of one hand. There was no paralysis that he could detect, and a very minimal amount of sensory disturbance, no thermo-anesthesia, yet her astereognosis was very pronounced, though confined to one hand.

Dr. Patrick added that a diagnosis of hysteria had been made by a very competent man, but after weeks of observation Dr. Patrick was convinced it was not a case of hysteria at all. There was only one conclusion to be reached; that wherever that woman's lesion is, it must be in such a place that it does not interfere with motion, but does interfere particularly with this peculiar, complicated, composite stereognosis, and interferes very little with the faculty of common sensation. With regard to astereognosis, it seemed to him that it may and should be produced by lesions variously located. It is a most complicated affair and the cutting of fibers running to a suppositional center from different and possibly widely separated regions of the brain could easily interfere with such a mechanism.

Dr. Joseph Sailer said that about a year and a half ago he studied a case of hemiplegia in which astereognosis was present, and he believed it was in that paper that the term astereognosis was first used. It occurred to him then and since, for he had seen a number of other cases in which the condition was present, that we cannot regard astereognosis as a sharp, distinct, localizing symptom, although in the majority of cases there seems to be a good deal of reason for accepting parietal localization. Dr. Sailer looked up as many cases as he could find, and had come across others under various names in the older literature, because the term stereognostic sense was not employed very extensively even after Hoffmann's paper, and even Oppenheim in '94 used the word "Tastsinn" to designate it, and other cases have been described under the same term. In some cases the parietal lobe was not involved, and there were lesions in other parts of the brain. Dr. Sailer saw a typical case of astereognosis last winter in a patient suffering with locomotor ataxia, in which all forms of sensation were normal in the part except a slight degree of disturbance of the muscular sense. He thought in a good many of the cases recorded there has not been a sufficiently careful study of the peripheral sensations that have been disturbed, because in a majority of cases when we have a loss of the tactile sensation—he thought Dr. Burr had placed one case on record in which it is not so—the stereognostic sense is necessarily lost. When other sensations are lost it may or may not be disturbed. There seems to be a curious irregularity about this. Dr. Dercum holds that the spacing sense, which is a term for a rather crude idea in psychology, is very important. Dr. Sailer believed that the localization sense combined with more or less disturbance of the muscular sense was a very important factor. Another point that ought to be well considered is the transient loss of stereognosis. He had reported a case with left hemiplegia which had persistent astereognosis in the left hand and transient astereognosis (for about thirty-six hours) in the right hand. In this case it might be due simply to the general cerebral disturbance and not to any focal lesion that would involve the fibers from the right hand to the brain, or the centers in the brain. He agreed with Dr. Patrick that what we need in the whole study is more facts and also more attention to the details of sensory phenomena as they occur in the different cases.

Dr. Hugh T. Patrick in regard to the practical applicability of this separate sensory area, said that some of the most brilliant localizations he had made had been made on the assumption that the post-Rolandic area was sensory. He must also say that some of the most brilliant errors he had made had been made by proceeding upon the same assumption. One case, not operated upon, seemed to be perfectly conclusive. She came to the necropsy table and a small tumor in the leg center (where of course it was supposed to be) was found, but not posterior to the Rolandic area, where it had been localized on account of sensory symptoms, but well to the front of it, and that tumor was not one which could well have produced symptoms at a distance. It was small, not larger than the end of the thumb, and infiltrating. There was no reason to believe that that tumor had any pressure effect. In fact, when the brain was cut it looked like a bruise, and a very competent pathologist who was present when the incision was made said, "I am not sure that it is a tumor at all." It was simply a localized discoloration, and there was no evidence that it exerted any pressure whatever on surrounding tissues.

Dr. Mills asked whether Dr. Patrick were sure there was no infiltration at all?

Dr. Patrick said that subsequent microscopic examination was made and the delimitations of the growth, a glioma, were very neat, but it had infiltrated the cortex, a cortical tumor, and extended but very little beneath the cortex. It began in the brain tissue, was not adherent to the membrane, and it was impossible to make out how it could have exerted any influence on the post-Rolandic region. It did not go deep. The whole growth was not bigger than the last phalanx of the thumb.

Dr. Joseph Collins asked Dr. Patrick to say whether or not there were in this case sensory phenomena and astereognosis.

Dr. Hugh T. Patrick did not know whether there was astereognosis or not. There were sensory symptoms and on account of them he had made the localization in the post-Rolandic region. He could not detail the sensory symptoms as this case occurred three or four years ago, and he had not refreshed his memory by consulting his notes. He simply stated the fact that it was an example of brilliant error in localization. The tumor was in the leg center in the front of the Rolandic region. It was wholly in front and not wholly behind where he had supposed it probably was.

Dr. Charles L. Dana said that when he first began the study of this subject, his conclusions were that the sensory zone overlaid the motor zone, and that they were pretty closely identical, though no single sensory zone or center had any such sharp definition as the motor centers had.

He still believed that the cutaneous and muscular sensations have a rather diffuse representation in the cortex of the brain, each particular part of the skin having quite a large center, and probably one in each hemisphere. He thought it is the general view of those who claim identity of motor and sensory centers that the latter extend perhaps into the prefrontal convolutions, and certainly back into the parietal lobe. He had always contended that muscular sensations were represented in the inferior parietal lobe.

Three weeks ago he had a case in which Dr. Murray removed a tumor from the inferior parietal region back of the posterior central convolution. The man had a slight amount of right hemiplegia; he had a little paresthesia of the hand and of the leg; absolutely no objective disturbance at all, no astereognosis, none of the loss of tactile or motor touch, and no distinct anesthesia. The tumor was a sarcoma infiltrating the inferior parietal lobe just behind the posterior central lobe. Some time ago a surgeon cut out practically the larger part of the paracentral lobule in a case of Jacksonian epilepsy. The patient made a good recovery surgically. There was no infiltration, and no extension of the disease, and the patient had hemiplegia most marked in the leg, and with it some hemianesthesia. Those are the old arguments Dr. Mills said he has not brought forward. It seemed to Dr. Dana as long as we find over and over again that cutting out a piece of the motor cortex produces in a certain percentage of cases anesthesia or a certain amount of anesthesia; we cannot deny that the motor cortex has something to do with the representation of cutaneous sensation.

With rgard to the definite localization of the center for stereognosis in the parietal lobe, in the analysis of the case he first investigated of lesions of the motor cortex and published in a paper about four or five years ago, he came to the conclusion that the center for motor touch was in the central convulution. The very earliest sign of disease of the central convolution was a little incapacity of the fingers, hand center, to appreciate the nature and locality of objects in them, and upon facts which he saw and collected regarding that point, he drew up the theory of a sort of aphasia of motion which it seemed to him is practically the same thing that was worked out later, which Dr. Dana thought Dr. Walton alluded to in his paper. At that time it seemed that the most characteristic sensory symptom of slight degree of lesion of the motor cortex was loss of this motor touch. If Dr. Mills denies that we shall have to continue the argument.

Dr. C. K. Mills said it was manifestly impossible for him to go over in detail the remarks which had been made in the discussion, not that the facts and real arguments against his position are many; they are exceedingly few. In the whole of this discussion the only two new facts which seemed to have any value are embodied in the case presented by Dr. Patrick and possibly that presented by Dr. Collins. As for Dr. Knapp (we are never personal in this Association) he had made a single contribution to this subject, which Dr. Mills thought could be explained otherwise than Dr. Knapp would explain it. His remarks on Dr. Mills' paper were simply to say something had been said which was not asserted. It is true Dr. Morton, of New York, at the last meeting of this Association, held in Boston, in a flippant and somewhat amusing manner said that Dr. Dana had led him (Dr. Mills) to water at that trough which is called the Rolandic fissure, or something of this sort. Dr. Mills did not acknowledge that he had brought his sensory area to that fissure, and he would defy Dr. Knapp to find anything on record which proves that he did. Even if he did this, still the main point of his contention remains that the largest part of the Rolandic area is now acknowledged by these gentlemen to be motor. Nothing to the contrary has been said. Dr. Knapp said something to the effect that we should in all the cases that had been examined for astereognosis have known their previous condition as regards sensation, etc. In Philadelphia Dr. Mills' colleagues, if not himself, do examine as to normal conditions when they have a chance to do it, to suggest anything else was a very trifling sort of argument to make against a position of the kind taken by Dr. Mills. Dr. Mills was glad that while it was asserted that he was led to this fissure, that he had been able to assist in leading two-thirds of the members of this Association to that motor area which he believed to be a purely motor area.

With regard to Dr. Collins he had given one case which may perhaps be disregarded because he had not been able to limit its subcortical extensions, at least as yet. Otherwise, as much as Dr. Mills admired his argumentative powers, really he saw nothing in his remarks but a repetition of statements as to his belief.

He could go over all the old arguments on this subject, giving from Shaefer or from his own book, the proper arguments in support of his position, but his object was chiefly to present four or five new points with regard to this matter. With reference to the remarks of the President, he should say that a single case may have very great value, many cases of the same sort of course having much more value. A single case thoroughly studied, of lesion of the motor cortex, is of great importance. Nothing was more striking to him in his discussion than the one case which Dr. Dercum and Dr. Spiller had presented to prove the existence of the *carrefour sensitif*. We have waited a score of years for that case, and now have it, and it is a good one. It is time for members of this Association to acknowledge facts; it is time we should stop simply collecting cases in which it happens that sensory and motor functions have been injured in the same case and which can be explained in nine out of ten cases by the fact that the sensory and motor areas are co-terminous. One area encircles the other; they may dentate into each other, because their frontiers are not like the lines of our diagrams. Dr. Mills said that in spite of ridicule, and in spite of the arguments which have been presented one year after another, he had gotten his brethren a little farther on the way.